

Fscn2 Cas9-CKO Strategy

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Project Overview



Project Name

Fscn2

Project type

Cas9-CKO

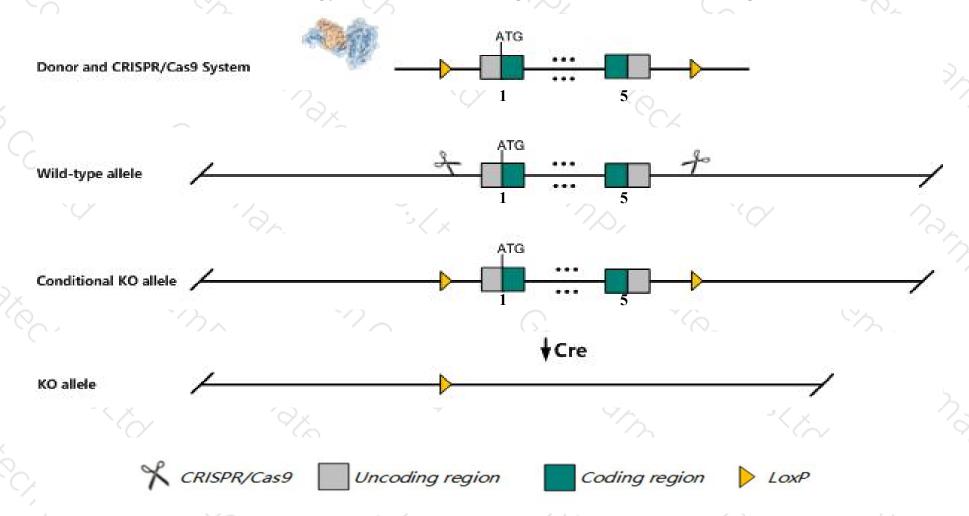
Strain background

C57BL/6JGpt

Conditional Knockout strategy



This model will use CRISPR/Cas9 technology to edit the Fscn2 gene. The schematic diagram is as follows:



Technical routes



- The *Fscn2* gene has 3 transcripts. According to the structure of *Fscn2* gene, exon1-exon5 of *Fscn2-201* (ENSMUST00000026445.2) transcript is recommended as the knockout region. The region contains all of the coding sequence. Knock out the region will result in disruption of protein function.
- ➤ In this project we use CRISPR/Cas9 technology to modify *Fscn2* gene. The brief process is as follows:CRISPR/Cas9 system and Donor were microinjected into the fertilized eggs of C57BL/6JGpt mice. Fertilized eggs were transplanted to obtain positive F0 mice which were confirmed by PCR and sequencing. A stable F1 generation mouse model was obtained by mating positive F0 generation mice with C57BL/6JGpt mice.
- The flox mice will be knocked out after mating with mice expressing Cre recombinase, resulting in the loss of function of the target gene in specific tissues and cell types.

Notice



- ➤ According to the existing MGI data, Mice homozygous for disruptions in this gene display retinal generation with structural abnormalities of the outer segment and depressed rod and cone ERGs that worsen with age.
- The floxed region is near to the C-terminal of *Faap100* gene, this strategy may influence the regulatory function of the C-terminal of *Faap100* gene.
- > The *Fscn2* gene is located on the Chr11. If the knockout mice are crossed with other mice strains to obtain double gene positive homozygous mouse offspring, please avoid the two genes on the same chromosome.
- ➤ This Strategy is designed based on genetic information in existing databases. Due to the complexity of biological processes, all risk of loxp insertion on gene transcription, RNA splicing and protein translation cannot be predicted at existing technological level.

Gene information (NCBI)



Fscn2 fascin actin-bundling protein 2 [Mus musculus (house mouse)]

Gene ID: 238021, updated on 3-Feb-2020

Summary

☆ ?

Official Symbol Fscn2 provided by MGI

Official Full Name fascin actin-bundling protein 2 provided by MGI

Primary source MGI:MGI:2443337

See related Ensembl:ENSMUSG00000025380

Gene type protein coding
RefSeq status VALIDATED
Organism Mus musculus

Lineage Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi; Mammalia; Eutheria; Euarchontoglires; Glires; Rodentia;

Myomorpha; Muroidea; Muridae; Murinae; Mus; Mus

Also known as Ahl8; A930022G03; C630046B20Rik

Expression Low expression observed in reference dataset <u>See more</u>

Orthologs <u>human</u> all

Genomic context



Location: 11; 11 E2

See Fscn2 in Genome Data Viewer

Exon count: 5

Annotation release	Status	Assembly	Chr	Location
108	current	GRCm38.p6 (GCF_000001635.26)	11	NC_000077.6 (120360165120368173)
Build 37.2	previous assembly	MGSCv37 (GCF_000001635.18)	11	NC_000077.5 (120222848120229487)

Transcript information (Ensembl)



The gene has 3 transcripts, all transcripts are shown below:

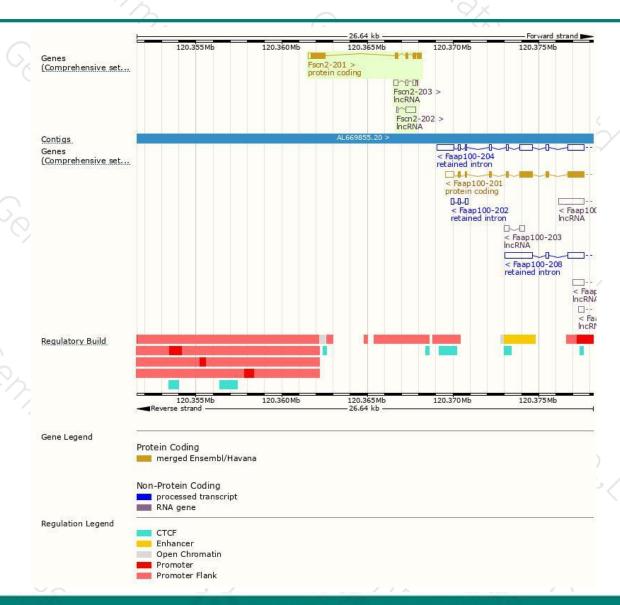
Name	Transcript ID	bp	Protein	Biotype	CCDS	UniProt	Flags
Fscn2-201	ENSMUST00000026445.2	1713	492aa	Protein coding	CCDS25731	Q32M02	TSL:1 GENCODE basic APPRIS P1
Fscn2-202	ENSMUST00000130476.1	669	No protein	IncRNA	9 4 3	6.00	TSL:3
Fscn2-203	ENSMUST00000152556.1	591	No protein	IncRNA	027	(40)	TSL:2

The strategy is based on the design of Fscn2-201 transcript, The transcription is shown below



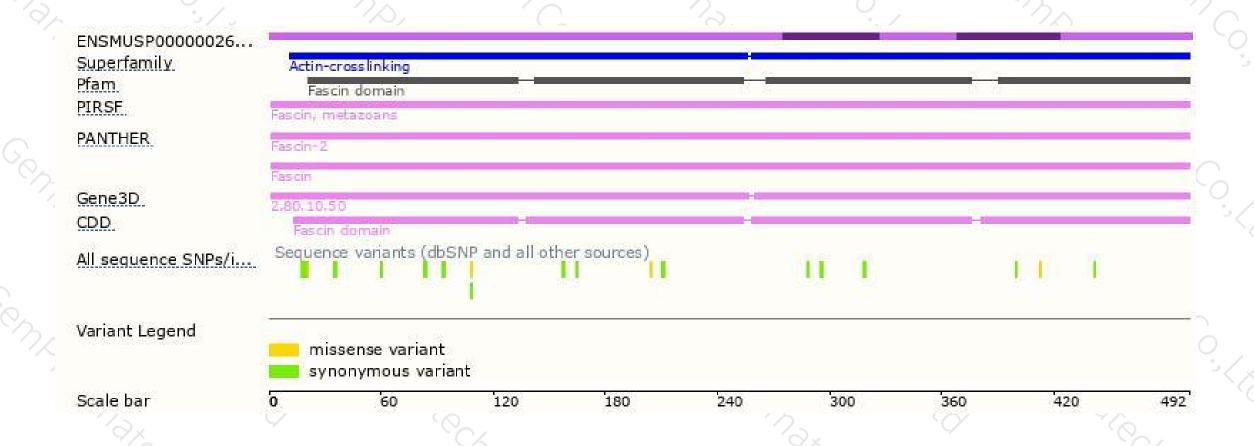
Genomic location distribution





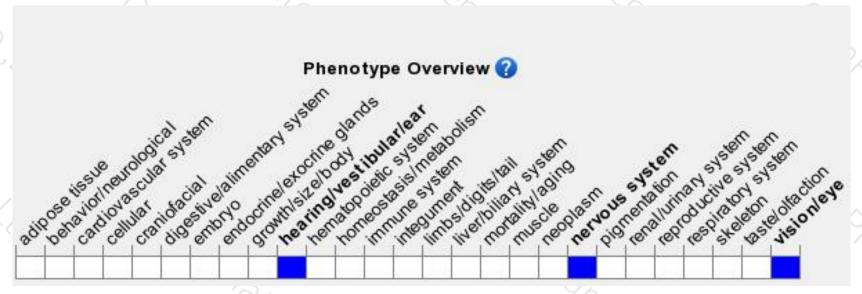
Protein domain





Mouse phenotype description(MGI)





Phenotypes affected by the gene are marked in blue.Data quoted from MGI database(http://www.informatics.jax.org/).

According to the existing MGI data, Mice homozygous for disruptions in this gene display retinal generation with structural abnormalities of the outer segment and depressed rod and cone ERGs that worsen with age.



If you have any questions, you are welcome to inquire. Tel: 400-9660890





